(FILE 'HOME' ENTERED AT 10:13:57 ON 18 FEB 2004)

FILE 'MEDLINE, CANCERLIT, BIOSIS, EMBASE, CAPLUS' ENTERED AT 10:14:21 ON 18 FEB 2004

L1	1922	S	ABCA#
L2	1163608	S	CHOLESTEROL OR LIPID
L3	1333	S	L1 AND L2
L4	743	S	GENE AND L3
L5	365	DUP REM	L4 (378 DUPLICATES REMOVED)
L6	29	S	L5 AND REVIEW
L7	15	S	ABC5 OR ABCA5
L8	10	DUP REM	L7 (5 DUPLICATES REMOVED)

L6 ANSWER 29 OF 29 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:834288 CAPLUS
DN 134:113806
TI Tangier's disease as a test of the reverse **cholesterol** transport hypothesis
AU Tall, Alan R.; Wang, Nan
CS Division of Molecular Medicine, Department of Medicine, Columbia University, New York, NY, 10032, USA
SO Journal of Clinical Investigation (2000), 106(10), 1205-1207
CODEN: JCINAO; ISSN: 0021-9738
PB American Society for Clinical Investigation
DT Journal; General Review
LA English
AB A **review** with 31 refs. The role of **ABCA1 gene** expression and HDL as a marker for **cholesterol** transport in pathogenesis of Tangier disease is discussed.
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 25 OF 29 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:715606 CAPLUS
DN 138:86925
TI **ABCA2**: a candidate regulator of neural transmembrane **lipid** transport
AU Schmitz, G.; Kaminski, W. E.
CS Institute for Clinical Chemistry and Laboratory Medicine, University of Regensburg, Regensburg, 93042, Germany
SO Cellular and Molecular Life Sciences (2002), 59(8), 1285-1295
CODEN: CMLSFI; ISSN: 1420-682X
PB Birkhaeuser Verlag
DT Journal; General Review
LA English
AB A **review**. Studies in the past years have implicated multispan transmembrane transport mols. of the ATP binding cassette (ABC) transporter family in cellular **lipid** export processes. The prototypic ABC transporter **ABCA1** has recently been demonstrated to act as a major facilitator of cellular **cholesterol** and phospholipid export. Moreover, the transporter **ABCA4** (ABCR) plays a pivotal role in retinaldehyde processing, and **ABCA3** has recently implicated in lung surfactant processing. These pioneering observations have directed considerable attention to the A subfamily of ABC proteins. **ABCA2** is the co-defining member of the ABC A-transporter subclass. Although known for some time, it was not until recently that its complete mol. structure was established. Unlike other ABC A-subfamily members, **ABCA2** is predominantly expressed in the brain and neural tissues. The unique expression profile together with available structural data suggest roles for this largest known ABC protein in neural transmembrane **lipid** export.

L6 ANSWER 23 OF 29 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:347378 CAPLUS
DN 139:50748
TI High-density lipoprotein subpopulations in pathologic conditions
AU Asztalos, Bela F.; Schaefer, Ernst J.
CS Atherosclerosis Research Laboratory, New England Medical Center, and the Lipid Metabolism Laboratory, Jean Mayer-United States Department of Agriculture/Human Nutritiion Research Center on Aging, Tufts University, Boston, MA, USA
SO American Journal of Cardiology (2003), 91(7A), 12E-17E
CODEN: AJCDAG; ISSN: 0002-9149
PB Excerpta Medica, Inc.
DT Journal; General Review
LA English
AB A **review**. The role of low-d. lipoprotein (LDL) **cholesterol** in coronary artery disease (CAD) and the impact of therapeutic agents on LDL **cholesterol** are well established. Less is known about the role of high-d. lipoprotein (HDL) **cholesterol** and even less about the role of the different HDL subspecies in CAD. HDL particles vary in size and d., mainly because of differences in the number of apolipoprotein (apo) particles and the amount of **cholesterol** ester in the core of HDL. Apo A-I is essential and, together with **lipid**, sufficient for the formation of HDL particles. Apo A-I-containing HDL particles play a primary role in **cholesterol** efflux from membranes, at least in part through interactions with the ATP-binding cassette transporter A1 (**ABCA1**). Patients with Tangier disease have mutations in the **gene** encoding **ABCA1**, which result in functionally impaired protein, a marked deficiency in HDL **cholesterol**, and a high risk of premature CAD. Our studies of apo A-I-containing HDL subpopulations in various patient populations reveal that patients homozygous for Tangier disease have only the pre-β1 HDL subspecies. Tangier heterozygotes are severely depleted in the larger α- and pre-α-mobility subspecies. Patients with low HDL **cholesterol** levels and those with CAD also show deficiencies in the α1 and pre-α1-3 HDL subspecies. The 3-hydroxy-3-methylglutaryl CoA reductase inhibitors (statins) increase the levels of the large α1 and pre-α1 subpopulations and decrease the level of the small α3 subpopulation. Thus, atorvastatin, for example, significantly moves the distribution of HDL particles toward normal, followed by simvastatin, pravastatin, and lovastatin in decreasing order of efficiency. A new statin, rosuvastatin, produces greater increases in HDL **cholesterol** than atorvastatin, but its effect on HDL particle distribution is yet to be determined

L6 ANSWER 21 OF 29 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AN 2000193470 EMBASE

TI **ABCA1**-mediated transport of cellular **cholesterol** and phospholipids to HBL apolipoproteins.

AU Oram J.F.; Vaughan A.M.

CS J.F. Oram, Department of Medicine, University of Washington, Box 356426, Seattle, WA 98195, United States. joram@u.washington.edu

SO Current Opinion in Lipidology, (2000) 11/3 (253-260).
Refs: 56
ISSN: 0957-9672 CODEN: COPLEU

CY United Kingdom

DT Journal; General Review

FS 018 Cardiovascular Diseases and Cardiovascular Surgery

LA English

SL English

AB **Lipid**-poor apolipoproteins remove cellular **cholesterol** and phospholipids by an active transport pathway controlled by an ATP binding cassette transporter called **ABCA1** (formerly ABC1). Mutations in **ABCA1** cause Tangier disease, a severe HDL deficiency syndrome characterized by a rapid turnover of plasma apolipoprotein A-I, accumulation of sterol in tissue macrophages, and prevalent atherosclerosis. This implies that lipidation of apolipoprotein A-I by the **ABCA1** pathway is required for generating HDL particles and clearing sterol from macrophages. Thus, the **ABCA1** pathway has become an important therapeutic target for mobilizing excess **cholesterol** from tissue macrophages and protecting against atherosclerosis.

L6 ANSWER 20 OF 29 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AN 2000360862 EMBASE

TI ABC transporters in cellular **lipid** trafficking.

AU Schmitz G.; Kaminski W.E.; Orso E.

CS Dr. G. Schmitz, Inst. for Clinical Chem./Lab. Med., University of Regensburg, Franz-Josef-Strauss-Allee 11, D-93042 Regensburg, Germany. gerd.schmitz@klinik.uni-regensburg.de

SO Current Opinion in Lipidology, (2000) 11/5 (493-501).
Refs: 75
ISSN: 0957-9672 CODEN: COPLEU

CY United Kingdom

DT Journal; General Review

FS 005 General Pathology and Pathological Anatomy
018 Cardiovascular Diseases and Cardiovascular Surgery
022 Human Genetics

LA English

SL English

AB ATP-binding cassette (ABC) transporters constitute a group of evolutionary highly conserved cellular transmembrane transport proteins. Recent work has implicated ABC transporters in cellular transmembrane **lipid** transport and hereditary diseases have been causatively linked to defective ABC transporters translocating **lipid** compounds. The emerging concept that a defined subset of ABC transporters is intimately involved in cellular **lipid** trafficking has recently been substantiated convincingly by the finding that **ABCA1** plays a central role in the regulation of HDL metabolism and macrophage targeting to the RES or the vascular wall. Differentiation dependent expression of a large number of ABC transporters in monocytes/macrophages and their regulation by sterol flux render these transporter molecules potentially critical players in atherogenesis and other chronic inflammatory diseases. (C) 2000 Lippincott Williams and Wilkins.

L6 ANSWER 18 OF 29 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AN 2001087277 EMBASE

TI Novel approaches to treating cardiovascular disease: Lessons from Tangier disease.

AU Oram J.F.

CS J.F. Oram, Department of Medicine, University of Washington, Box 356426, Seattle, WA 98195-6426, United States. joram@u.washington.edu

SO Expert Opinion on Investigational Drugs, (2001) 10/3 (427-438).
Refs: 76
ISSN: 1354-3784 CODEN: EOIDER

CY United Kingdom

DT Journal; General Review

FS 005 General Pathology and Pathological Anatomy
018 Cardiovascular Diseases and Cardiovascular Surgery
022 Human Genetics
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index

LA English

SL English

AB Atherosclerotic cardiovascular disease (CVD) remains the leading cause of morbidity and mortality in Western societies. Although **cholesterol** is a major CVD risk factor, therapeutic interventions to lower plasma **cholesterol** levels have had limited success in reducing coronary events. Thus, novel approaches are needed to reduce or eliminate CVD. A potential therapeutic target is a newly discovered ATP binding cassette transporter called **ABCA1**, a cell membrane protein that is the gateway for secretion of excess **cholesterol** from macrophages into the high density lipoprotein (HDL) metabolic pathway. Mutations in **ABCA1** cause Tangier disease, a severe HDL deficiency syndrome characterised by accumulation of **cholesterol** in tissue macrophages and prevalent atherosclerosis. Studies of Tangier disease heterozygotes revealed that the relative activity of **ABCA1** determines plasma HDL levels and susceptibility to CVD. Drugs that induce **ABCA1** in mice increase clearance of **cholesterol** from tissues and inhibit intestinal absorption of dietary **cholesterol** . Thus, **ABCA1**-stimulating drugs have the potential to both mobilise **cholesterol** from atherosclerotic lesions and eliminate **cholesterol** from the body. By reducing plaque formation and rupture independently of the atherogenic factors involved, these drugs would be powerful agents for treating CVD.

L6 ANSWER 16 OF 29 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AN 2002158514 EMBASE

TI **ABCA1**: Regulation, function and relationship to atherosclerosis.

AU Francone O.L.; Aiello R.J.

CS O.L. Francone, Pfizer Global Res. and Development, Eastern Point Road, Groton, CT 06340, United States. omar_l_francone@groton.pfizer.com

SO Current Opinion in Investigational Drugs, (2002) 3/3 (415-419).
Refs: 42
ISSN: 1472-4472 CODEN: CIDREE

CY United Kingdom

DT Journal; General Review

FS 018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
037 Drug Literature Index
030 Pharmacology

LA English

SL English

AB Since the identification of mutations in the ATP-binding cassette transporter (**ABCA1**), the relationship between **ABCA1** expression, **cholesterol** efflux, high-density lipoprotein (HDL) biosynthesis and **cholesterol** homeostasis has been a subject of intense investigation. Several studies have provided significant new information with regards to pathways controlling **ABCA1** expression and activity and established that this transporter facilitates the efflux of **cholesterol** and phospholipids to apoprotein acceptors, leading to the formation of nascent HDL particles. Although **ABCA1** appears to play a critical role in **cholesterol** flux from tissues, and despite a considerable interest in developing pharmacological agents that increase **ABCA1** activity, the role of **ABCA1** in preventing atherosclerosis remains unclear.

L6 ANSWER 15 OF 29 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AN 2002174459 EMBASE

TI Nuclear hormone receptors and **cholesterol** trafficking: The orphans find a new home.

AU Fitzgerald M.L.; Moore K.J.; Freeman M.W.

CS M.W. Freeman, Department of Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA 02114, United States. Freeman@molbio.mgh.harvard.edu

SO Journal of Molecular Medicine, (2002) 80/5 (271-281).
Refs: 70
ISSN: 0946-2716 CODEN: JMLME8

CY Germany

DT Journal; General Review

FS 003 Endocrinology
006 Internal Medicine
029 Clinical Biochemistry

LA English

SL English

AB There are many homeostatic mechanisms that contribute to the regulation of cellular and serum **cholesterol** levels in humans. Much of our understanding of this regulation arose from studies of the cellular pathways controlling **cholesterol** synthesis and the uptake and degradation of low-density lipoproteins. The physiology governing **cholesterol** disposition in whole animals, including the molecular mechanisms responsible for dietary uptake of **cholesterol** and its subsequent catabolism to bile acids, are only now being uncovered. This **review** summarizes recent studies that have used modern genetic techniques, as well as cell biological methods, to begin to elucidate the pathways responsible for **cholesterol** trafficking in vivo. This work has led to the realization that networks of nuclear hormone receptors, including the LXR, FXR, PPAR, and RXR proteins, play critical roles in **lipid** metabolism by virtue of their transcriptional regulation of the genes that control sterol metabolic pathways. Some of the major downstream targets of these regulatory networks involve members of the ABC transporter family, including **ABCA1**, ABCG1, ABCG5, ABCG8, MDR3/Mdr2, and SPGP/BSEP. These transporters contribute to the movement of sterols and biliary lipids across cellular membranes via mechanisms that have yet to be elucidated. The potential for manipulating these **cholesterol** trafficking pathways via drugs targeted to the nuclear hormone receptors has generated great interest in their biology and will undoubtedly lead to new therapeutic approaches to human disorders in the coming years.

L6 ANSWER 11 OF 29 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:409910 BIOSIS
DN PREV200100409910
TI The human ATP-binding cassette (ABC) transporter superfamily.
AU Dean, Michael [Reprint author]; Rzhetsky, Andrey; Allikmets, Rando
CS Human Genetics Section, Laboratory of Genomic Diversity, National Cancer Institute-Frederick, Frederick, MD, 21702, USA
dean@ncifrcrf.gov
SO Genome Research, (July, 2001) Vol. 11, No. 7, pp. 1156-1166. print.
ISSN: 1088-9051.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 29 Aug 2001
Last Updated on STN: 22 Feb 2002
AB The ATP-binding cassette (ABC) transporter superfamily contains membrane proteins that translocate a variety of substrates across extra- and intra-cellular membranes. Genetic variation in these genes is the cause of or contributor to a wide variety of human disorders with Mendelian and complex inheritance, including cystic fibrosis, neurological disease, retinal degeneration, **cholesterol** and bile transport defects, anemia, and drug response. Conservation of the ATP-binding domains of these genes has allowed the identification of new members of the superfamily based on nucleotide and protein sequence homology. Phylogenetic analysis is used to divide all 48 known ABC transporters into seven distinct subfamilies of proteins. For each **gene**, the precise map location on human chromosomes, expression data, and localization within the superfamily has been determined. These data allow predictions to be made as to potential functions or disease phenotypes associated with each protein. In this paper, we **review** the current state of knowledge on all human ABC genes in inherited disease and drug resistance. In addition, the availability of the complete Drosophila genome sequence allows the comparison of the known human ABC genes with those in the fly genome. The combined data enable an evolutionary analysis of the superfamily. Complete characterization of all ABC from the human genome and from model organisms will lead to important insights into the physiology and the molecular basis of many human disorders.

L6 ANSWER 5 OF 29 MEDLINE on STN
AN 2002677228 MEDLINE
DN 22324858 PubMed ID: 12437993
TI Adenosine triphosphate-binding cassette transporter genes in ageing and age-related diseases.
AU Efferth Thomas
CS Virtual Campus Rhineland-Palatinate, Rodeneck Platz 2, 55126, Mainz, Germany.. efferth@vcrp.de
SO Ageing Res Rev, (2003 Jan) 2 (1) 11-24. Ref: 150
Journal code: 101128963. ISSN: 1568-1637.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200306
ED Entered STN: 20021120
Last Updated on STN: 20030605
Entered Medline: 20030604
AB The family of adenosine triphosphate (ATP)-binding cassette (ABC) transporters is the largest **gene** family known. While some ABC transporters translocate single substances across membranes with high specificity, others transport a wide variety of different lipophilic compounds. They are responsible for many physiological processes and are also implicated in a number of diseases. The present **review** focuses on ABC transporter genes which are involved in ageing and age-related diseases. Expression of ABCB1 (MDR1, P-glycoprotein) increases with age in CD4(+) and CD8(+) T-lymphocytes indicating that P-glycoprotein may be involved in the secretion of cytokines, growth factors, and cytotoxic molecules. As T cells in aged individuals are hyporesponsive leading to a reduced immunodefence capability, a role of ABCB1 in age-related immunological processes is presumed. The **ABCA1** (ABC1) **gene** product translocates intracellular **cholesterol** and phospholipids out of macrophages. Genetic aberrations in **ABCA1** cause perturbations in lipoprotein metabolism and contribute to atherosclerosis. **ABCA4** (ABCR) represents a retina-specific ABC transporter expressed in rod photoreceptor cells. The **ABCA4 gene** product translocates retinyl-derivatives. Mutations in the **ABCA4 gene** contribute to age-related macular degeneration. Polymorphisms in the sulfonylurea receptor **gene** (ABCC8, SUR1) are associated with non-insulin-dependent diabetes mellitus (NIDDM). Sulfonylureas inhibit potassium conductance and are used to treat NIDDM by stimulation of insulin secretion across ATP-sensitive potassium channels in pancreatic beta-cell membranes. Possible diagnostic and therapeutic implications of ABC transporters for age-related diseases are discussed.

L6 ANSWER 4 OF 29 MEDLINE on STN
AN 2003036217 MEDLINE
DN PubMed ID: 12544659
TI ATP-binding cassette transporter A1, fatty acids, and **cholesterol** absorption.
AU Brousseau Margaret E
CS Tufts University School of Medicine, and Lipid Metabolism Laboratory, Jean Mayer-USDA Human Nutrition Research Center on Aging, Boston, Massachusetts 02111, USA.. margaret.brousseau@tufts.edu
SO Current opinion in lipidology, (2003 Feb) 14 (1) 35-40. Ref: 52
Journal code: 9010000. ISSN: 0957-9672.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200308
ED Entered STN: 20030125
Last Updated on STN: 20030828
Entered Medline: 20030827
AB PURPOSE OF **REVIEW**: A significant advance in our understanding of the reverse **cholesterol** transport pathway occurred in 1999 with the identification of defects in the ATP-binding cassette transporter A1 **gene** as the cause of Tangier disease. Since this discovery, an overwhelming number of experiments have been conducted to further define the function of this **gene**. Among the concepts emerging from such studies is a possible role for the **gene** in **cholesterol** absorption. The present **review** summarizes the most recent of these studies, as well as the only report to describe the effects of fatty acids on ATP-binding cassette transporter A1 **gene** activity. RECENT FINDINGS: From the one study conducted thus far, it appears that unsaturated fatty acids can reduce ATP-binding cassette transporter A1 **gene** activity by enhancing its degradation. Among the primary modulators of the **gene**'s transcription is the liver X receptor, with liver X receptor-selective agonists significantly increasing expression of the **gene**. While some studies indicate that upregulation of the **gene** inhibits **cholesterol** absorption, the results of other studies suggest that it facilitates **cholesterol** absorption and the transfer of **cholesterol** into the bile. Preliminary evidence from studies with transgenic and knockout mice supports the concept that increasing ATP-binding cassette transporter A1 **gene** expression may be beneficial in the prevention of diet-induced atherosclerosis. SUMMARY: Although there is substantial evidence from and studies to suggest that the ATP-binding cassette transporter A1 **gene** regulates intestinal **cholesterol** absorption, perhaps by mediating **cholesterol** efflux from the basolateral surface of enterocytes, it remains unclear whether or not this **gene** is the primary ATP-binding cassette transporter involved in the process.

L8 ANSWER 9 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:324630 BIOSIS
DN PREV200300324630
TI EXPRESSION OF ABCA TRANSPORTER AT RAT AND HUMAN BLOOD - BRAIN BARRIER.
AU Ohtsuki, S. [Reprint Author]; Watanabe, Y. [Reprint Author]; Kamoi, M. [Reprint Author]; Kamiya, N. [Reprint Author]; Hori, S. [Reprint Author]; Terasaki, T. [Reprint Author]
CS Grad. Sch. of Pharm. Sci., NICHe, Tohoku Univ., Sendai, Japan
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 580.18. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 16 Jul 2003
Last Updated on STN: 16 Jul 2003
AB ABCA family belongs to ATP binding cassette (ABC) transporter superfamily and contains subtypes which are expressed in the brain and involved in the efflux transport system. Since the resent study has reported ABCB1 (MDR1) is not expressed at human blood-brain barrier (BBB), but in astrocyte foot processes1, it is important to clarify the BBB expression of other ABCA families in human. The purpose of this study is investigating the expression of ABCA family at rat and human BBB. The expression of ABCA1, 2, 3, 5 and 6 mRNA were detected in conditionally immortalized rat brain capillary cell lines (TR-BBB11 and/or 13) by RT-PCR. Further studies were performed about ABCA2 and 5, and those transporters mRNA were also detected in rat brain capillary fraction. In situ hybridization analysis exhibited that **ABCA5** was localized at cerebral cortex, hippocampus, lateral ventricles and cerebellum. These results suggest that ABCA2, 5 were expressed at rat BBB, and **ABCA5** was also expressed at rat blood-cerebrospinal fluid barrier. The mRNA expression in human brain was examined by northern blot analysis. ABCA2 were detected in all examined brain region at 8.1 kb. **ABCA5** were expressed in cerebellum intensely and also detected in cerebral cortex, occipital pole and frontal lobe at 7.1 kb. Furthermore, ABCA2 and 5 were detected in cultured human brain capillary endothelial cells by RT-PCR. This result suggests that ABCA2 and 5 are expressed at human BBB. ABCA2 and 5 could be involved in efflux transport system at rat and human BBB.
1. Pardridge W.M. et al. J. Neurochem 68,1278-1285 (1997).

L8 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:446159 CAPLUS
DN 137:29064
TI Human **ABCA5**, ABCA6, ABCA9, and ABCA10 genes encoding ATP binding cassette transporters and their use in diagnosis and treatment of diseases associated with reverse transport of cholesterol
IN Denefle, Patrice; Rosier-Montus, Marie-Francoise; Prades, Catherine; Arnoud-Reguigne, Isabelle; Duverger, Nicolas; Allikmets, Rando; Dean, Michael
PA Aventis Pharma S.A., Fr.
SO Eur. Pat. Appl., 142 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 1213352	A1	20020612	EP 2000-403440	20001207
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	WO 2002046458	A2	20020613	WO 2001-EP15401	20011207
	WO 2002046458	A3	20040108		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2002017166	A5	20020618	AU 2002-17166	20011207
	US 2003044895	A1	20030306	US 2001-5338	20011207
PRAI	EP 2000-403440	A	20001207		
	US 2001-263231P	P	20010123		
	WO 2001-EP15401	W	20011207		

AB The present invention relates to nucleic acids corresponding to various exons of **ABCA5**, ABCA6, ABCA9, and ABCA10 genes as well as cDNAs encoding the novel full length of **ABCA5**, ABCA6, ABCA9, and ABCA10 ATP binding cassette transporters. The invention also relates to means for the detection of polymorphisms in general, and of mutations in particular, in the **ABCA5**, ABCA6, ABCA9, and ABCA10 genes or in the corresponding protein produced by the allelic form of the **ABCA5**, ABCA6, ABCA9, and ABCA10 genes.
RE.CNT 11 THERE ARE 11 CITED REFERENCES

L8 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:674680 CAPLUS
DN 137:212031
TI Protein and cDNA sequences of human ATP-binding cassette transporter **ABCA5** and their uses in diagnosis and therapy
IN Chen, Hongyun; Kilinski, Ligia; Le, Bihan Stephane
PA Active Pass Pharmaceuticals, Inc., Can.
SO U.S. Pat. Appl. Publ., 52 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002123107	A1	20020905	US 2002-90458	20020301
WO 2002070690	A2	20020912	WO 2002-CA266	20020301
WO 2002070690	A3	20030116		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-272885P P 20010302
AB The invention provides human isolated nucleic acid mols., designated **ABCA5** transporter nucleic acid mols., which encode novel ABC transporter family members. The invention also provides antisense nucleic acid mols., recombinant expression vectors containing **ABCA5** transporter nucleic acid mols., host cells into which the expression vectors have been introduced, and non-human transgenic sterile animals in which an **ABCA5** transporter gene has been disrupted. The invention further provides isolated **ABCA5** transporter proteins, anti-**ABCA5** transporter antibodies, and screening assays for **ABCA5** transporter modulators. Diagnostic and therapeutic methods utilizing compns. of the invention are also provided.

L8 ANSWER 3 OF 10 MEDLINE on STN DUPLICATE 2
AN 2003025327 MEDLINE
DN 22419899 PubMed ID: 12532264
TI Evolutionary analysis of a cluster of ATP-binding cassette (ABC) genes.
AU Annilo Tarmo; Chen Zhang-Qun; Shulenin Sergey; Dean Michael
CS Human Genetics Section, Laboratory of Genomic Diversity, NCI-Frederick, Frederick, MD 21702, USA.
NC N01-C0-5600
SO MAMMALIAN GENOME, (2003 Jan) 14 (1) 7-20.
Journal code: 9100916. ISSN: 0938-8990.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF491299; GENBANK-AF491842; GENBANK-AF498360; GENBANK-AF498361; GENBANK-AF498362
EM 200306
ED Entered STN: 20030118
Last Updated on STN: 20030626
Entered Medline: 20030625
AB To study the evolutionary history of ATP-binding cassette (ABC) transporters in mammals, we have characterized a cluster of five ABCA-subfamily genes localized on mouse Chromosome (Chr) 11. The genes, named **Abca5**, Abca6, Abca8a, Abca8b, and Abca9, are arranged in a head-to-tail fashion in a cluster that spans about 400 kb of the genomic DNA, each gene occupying about 70 kb. The transcripts of these genes contain an open reading frame from 4863 (for Abca8a and Abca8b) to 4929 (for **Abca5**) nucleotides, and have distinct tissue-specific expression pattern. The predicted proteins contain two transmembrane domains and two nucleotide binding domains, arranged similar to the other members of ABCA subfamily. Similarity of both the genomic organization and primary structure among the genes in this cluster suggests that the duplications generating the cluster occurred relatively recently compared with most of the ABC genes in present-day mammalian genomes. For instance, the Fugu rubripes genome contains an ortholog for only one gene, **Abca5**, from this cluster. Phylogenetic and comparative sequence analysis reveals that after the divergence of rodent and primate lineages, at least one gene has been lost in each genome. In addition, we found that both mouse and human clusters show evidence of a number of gene conversions, in several cases involving intron sequences.

L8 ANSWER 1 OF 10 MEDLINE on STN DUPLICATE 1
AN 2002741174 MEDLINE
DN 22392638 PubMed ID: 12504089
TI Cloning of human and rat **ABCA5**/**Abca5** and detection of a human splice variant.
AU Petry Frauke; Kotthaus Andre; Hirsch-Ernst Karen I
CS Institute of Pharmacology and Toxicology, Department of Toxicology, University of Gottingen, Robert-Koch-Strasse 40, D-37075 Gottingen, Germany.
SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (2003 Jan 10) 300 (2) 343-50.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200302
ED Entered STN: 20021231
Last Updated on STN: 20030226
Entered Medline: 20030225
AB We presently report the cloning of cDNA sequences encoding the novel rat ATP-binding cassette (ABC) transporter **Abca5** and the orthologous human transporter, recently designated as **ABCA5**. Furthermore, the existence of a novel non-translated exon of the **ABCA5** gene, previously assigned to an ABCA gene cluster in the chromosomal region 17q24.2-3, is demonstrated. **Abca5** and **ABCA5** cDNAs are predicted to give rise to proteins of 1642 amino acids which exhibit the typical domain arrangement of ABC full transporters and share 90% identity within the amino acid sequences. A cDNA representing an **ABCA5** mRNA splice variant was cloned which would result in a truncated protein equivalent to an ABC half transporter. Northern blot analyses revealed expression of **ABCA5** or **Abca5** mRNA in several tissues, but particularly high **Abca5** mRNA expression was observed in rat testis. Up-regulation of **Abca5** mRNA expression during culture of primary rat hepatocytes suggests that hepatocyte cultures should provide a basis for investigation of **Abca5** gene regulation and elucidation of **Abca5** function.